

Complete Uterine Inversion Following Fundal Pressure During Placental Delivery at Home: A Case Report of Massive Postpartum Haemorrhage Requiring Blood Transfusion

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Complete Uterine Inversion; Postpartum Haemorrhage; Fundal Pressure; home Delivery; blood transfusion; third stage of labour, Obstetrics emergency; Maternal near-miss.

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ABSTRACT

Background: Complete uterine inversion is a rare but life-threatening obstetric emergency associated with severe postpartum haemorrhage, haemodynamic instability, and maternal mortality if not recognized and managed promptly. It most commonly occurs during the third stage of labour and may be precipitated by inappropriate obstetric interventions such as excessive cord traction or fundal pressure. Cases occurring after home delivery remain uncommon but carry a high risk of adverse maternal outcomes due to delayed recognition and referral.

We report the case of a 30-year-old un-booked multiparous woman who developed complete uterine inversion following the application of fundal pressure during placental delivery at a home birth attended by an unskilled birth attendant. She presented to our facility with profuse vaginal bleeding, severe lower abdominal pain, haemodynamic instability, and a protruding uterine mass consistent with complete uterine inversion. Following prompt resuscitation with intravenous fluids, uterotonics after uterine replacement, and three (3) units of compatible whole blood, successful manual repositioning of the uterus was achieved using the Johnson's technique. The patient recovered without major complications and was discharged home in stable condition on the fourth post-admission day. Other techniques that can be used if Manual replacement fails are Huntington's/O'Sullivan's techniques.

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INTRODUCTION

Acute puerperal uterine inversion is a rare but catastrophic obstetric emergency in which the uterine fundus collapses into the endometrial cavity and may protrude through the cervix or vagina, usually during the third stage of labour. Its reported incidence ranges from approximately 1 in 2,000 to 1 in 50,000 deliveries, reflecting differences in obstetric practice and case ascertainment.¹⁻⁴ Despite its rarity, uterine inversion remains an important cause of severe postpartum haemorrhage (PPH), haemorrhagic shock, and preventable maternal mortality, particularly in low-resource settings where delayed recognition and referral are common.^(1,3,5) The condition is frequently associated with inappropriate management of the third stage of labour, including excessive umbilical cord traction before placental separation, inappropriate fundal pressure, uterine atony, morbidly adherent placenta, short umbilical cord, and fundal placental implantation.^{1,4,6-9} The diagnosis is primarily clinical and is characterized by sudden postpartum haemorrhage, severe pelvic pain, haemodynamic instability that may be disproportionate to the observed blood loss because of a

neurogenic component, and the absence of the uterine fundus on abdominal examination with or without a protruding vaginal mass.(1,3,5,10)

Management requires immediate multidisciplinary intervention comprising aggressive maternal resuscitation, prompt uterine replacement, administration of uterotonic agents following successful repositioning, correction of hypovolaemia with blood products, and treatment of associated coagulopathy.(1,5,11–13) Early recognition and adherence to evidence-based management protocols are associated with excellent maternal outcomes, whereas delays, particularly after home deliveries conducted by unskilled birth attendants, substantially increase morbidity and mortality.(7,12,14,15)

CASE PRESENTATION

A 30-year-old un-booked P5+0 (5A) Kanuri woman who had vaginal delivery of a live female neonate at term that cried immediately at birth about one (1) hour prior to presentation to our facility. The delivery was conducted at home by a traditional birth attendant (TBA) and presented to the obstetrics triage with an hour history of dizziness and fleshy vaginal protrusion and was managed as a case of complete uterine inversion following fundal pressure during placental delivery at home, complicated by massive postpartum haemorrhage requiring blood transfusion. All her previous deliveries were at home. There is no previous history of postpartum haemorrhage. No previous history of caesarean section or uterine surgery. However, she bled torrentially after the uterus became inverted and the TBA detached the placenta from the placental bed. On examination: she was conscious and alert but in painful distress, pale, afebrile (Temperature 37.1 degrees Celsius), not jaundiced, not cyanosed, dehydrated. Pulse rate=102 bpm regular, full volume, BP=130/70mmHg, HS=S1 & S2 only, SPO2=98% in room air, bedside urinalysis was negative for Glucose and protein. Abdomen was full, moved with respiration, liver and spleen not palpably enlarged, kidneys were not ballot- able, uterus not palpable per abdomen. Urethral Foley's catheter passed drained 200ml of concentrated urine.

Pelvic examination revealed: Bulky reddish looking vaginal mass (FIG 1), and her perineum and wrapper blood stained with blood, there was blood oozing from the placental bed of the inverted uterus. The RR=22 cpm, good air entry bilaterally.

Investigation results revealed: urgent packed cell volume of 24% (moderate anaemia), Haemoglobin=8.5g/dL, reduced RBC of $3.0 \times 10^6/\mu\text{L}$, MCV of 86 fL (Normocytic), Normal MCH of 28 pg, Normal MCHC of 33g/dL, WBC showed mild postpartum leucocytosis of $13.0 \times 10^9/\text{L}$, Platelets= $210 \times 10^9/\text{L}$, blood group O positive, Hb Genotype AA. EUCR essentially normal: Sodium 138mmol/L, Potassium 4.1mmol/L, Chloride 103mmol/L, Bicarbonate 24mmol/L. Bedside urinalysis was negative for Glucose and protein. Random Blood sugar of 6.5mmol/L. Coagulation profile: PT 12.5 seconds, INR 1.0, aPTT 30 seconds and fibrinogen 4.0 g/L. peripheral blood film showed Normocytic, normochromic red cells with mild polychromatic cells consistent with recent blood loss. HBsAg, HCV and retroviral screening were Non –Reactive.

The treatment of this patient with acute uterine inversion complicated by primary postpartum haemorrhage is an obstetrics emergency which involved simultaneous resuscitation and immediate correction of the inversion. The managing team immediately called for help including the senior obstetrician, midwives/nurses and blood bank immediately, while the anaesthetist and the theatre team also alerted. Immediate resuscitation according to the departmental protocol commenced by following the A, B, C Approach. Two large- bore IV cannulas were used to secure intravenous access and 1L of 0.9% Normal saline rushed fast. Blood samples for the above investigations were taken with results as stated above. Urinary Foley's catheter passed to monitor input/output, blood also send for grouping and cross-matching of 3units of whole blood. She was also administered intramuscular Diclofenac 75mg stat for pain and IV Metronidazole 500mg stat, Iv Ceftriaxone 1 g stat for prophylaxis against infections. With the patient in lithotomy position and using a gloved hand, the inverted uterine fundus was grasped and a steady upward pressure directing the inverted uterus through the cervical ring and back toward its normal anatomical position with the pressure being directed toward the umbilicus. The uterine hand was maintained in position until uterine contractions began following the commencement of another 1L of 0.9% Normal saline with 20 IU Oxytocin infusion was commenced and ran for 4 hours producing firm uterine contractions as uterine massage was also provided.. The manual reduction was successfully done (Johnson Manoeuvre) as shown in FIG. 2 below. Intravenous Tranexamic acid 1g in 10ml solution was given slowly over 10mins, 1000ug Misoprostol tablets passed per rectum, the genital tract examined for lacerations and found to be intact. Vital signs were closely monitored which remained stable and she was later transferred to the postnatal ward for further observations. She was transfused with 3 units of whole blood under intravenous frusemide cover 40mg before and after each transfusion. Her post transfusion packed cell volume was 34%.

She was further prescribed Tabs Metronidazole 400mg t d s x5/7, Caps Amoxicillin 500mg t d s x5/7, Tabs paracetamol 1 g t d s x 3/7, Tabs ferrous sulphate 60mg b. d x 6/52, tabs Folic acid 5mg daily x 6/52, Tabs Ascorbic acid 200mg t d s x 14/7. She was further monitored and was later discharged home to continue her postnatal follow up at the postnatal clinic.



FIG 1: picture showing Acute Uterine inversion at presentation



FIG 2: Picture of Acute uterine inversion after Manual reduction

DISCUSSION

Acute puerperal uterine inversion is an uncommon but life-threatening obstetric emergency that requires immediate diagnosis and intervention to prevent maternal mortality. Although the reported incidence varies considerably, it remains one of the most dramatic causes of postpartum haemorrhage (PPH) and haemorrhagic shock because inversion prevents effective uterine contraction and compression of the placental bed. (16–18). In the present case, complete uterine inversion occurred following the application of fundal pressure during placental delivery at home, a recognised but preventable precipitating factor associated with inappropriate management of the third stage of labour. (16, 19)

Current evidence indicates that excessive fundal pressure and premature controlled cord traction before placental separation are the most important iatrogenic risk factors, particularly in settings where deliveries are conducted by unskilled birth attendants. (16,19,20) Similar to previously reported cases, our patient presented with massive PPH, haemodynamic instability and complete uterine prolapse, necessitating prompt resuscitation, uterine replacement and blood transfusion. (18,20,21) Early restoration of circulating volume, manual replacement of the uterus before oedema develops, followed by uterotonic administration after successful repositioning, remain the cornerstone of management

This case also highlights the ongoing burden of preventable obstetric emergencies in low-resource settings, where home delivery and delayed referral continue to contribute substantially to maternal morbidity. Adherence to evidence-based management of the third stage of labour, avoidance of inappropriate fundal pressure, availability of emergency obstetric care and timely access to blood transfusion services are essential to reducing adverse outcomes. (19, 22–25)

Our case reinforces the need for continuous training of skilled birth attendants, community education regarding institutional delivery and prompt referral pathways for obstetric emergencies, particularly in resource-limited settings.

CONCLUSION

Complete uterine inversion is a rare but preventable obstetric emergency. This case demonstrates that inappropriate fundal pressure during placental delivery can precipitate catastrophic postpartum haemorrhage. Early recognition, prompt uterine replacement, aggressive resuscitation, timely blood transfusion, and skilled birth attendance are critical to preventing maternal morbidity and mortality.

PATIENT CONSENT

Written informed consent was obtained from the patient for publication of this case and the accompanying data. Patient identifiers have been removed.

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